EXHIBIT 18

UNITED STATES DISTRICT COURT

SOUTHERN DISTRICT OF NEW YORK

IN RE: Acetaminophen – ASD–ADHD Products Liability Litigation	**22-md-3043 (DLC)**
This Document Relates To: All Actions	

RULE 26 REBUTTAL EXPERT REPORT OF BRANDON PEARSON, MS, PhD

I have reviewed Defendants' expert reports dated July 21, 2023, and provide the following rebuttal report pursuant to Federal Rule of Civil Procedure 26. All opinions stated herein are offered to a reasonable degree of scientific certainty.

This rebuttal report responds to several assertions in the report of Craig M. Powell, MD, PhD, but it is not intended to be a comprehensive response to his opinions. My affirmative report, incorporated by reference herein, contains my response to many of the arguments raised in Dr. Powell's report. This rebuttal report identifies and responds to a sample of Dr. Powell's most egregious scientific errors and inaccuracies, which fundamentally undermine the reliability of his opinions in this case. In the event any new, relevant studies or information are published, obtained, or presented following submission of this rebuttal report, I reserve the right to review any such data and revise or supplement this report and my opinions accordingly.

"As long as we do not permit controlled experiments where we would intentionally harm a human subject, when there are no possible benefits to them, for the mere sake of scientific inquiry, no single experiment can provide decisive data on the effects of a foreign substance on a human group. In so far as we depend on a number of experiments, some with greater statistical or explanatory power than others and information from diverse forms of evidence, we need to have some way of aggregating or weighing the results across different modalities of evidence" (Krimsky 2005). In his report, Dr. Powell states that he evaluated the evidence using a systematic review methodology (Powell Report at paragraph 60). "A systematic review has been defined as a review of a clearly formulated question that uses systematic and explicit methods to identify, select, and critically appraise relevant research, and to collect and analyze data from the studies that are included in the review" (Avey et al., 2015).

In systematic review, as in the Weight of Evidence (WoE) approach I utilized, transparency is an essential requirement that ensures all interested parties have the opportunity to understand, reproduce, and question the analysis (Sena et al., 2014) ("In a systematic review, the researcher is required to outline aims, objectives, and methodology. The principle is that an independent researcher could perform the same identification process and yield the same data set. As is often the case, in the interpretation of primary research studies, the conclusions drawn from a meta-analysis may differ from one reviewer to the next. However, the transparency and objectivity of the techniques used provide a framework for these discussions"); (OECD 2019) ("The ultimate

goal of WoE is to provide a transparent means for communicating decision-making such that decisions can be clearly understood and questioned by all stakeholders"). Dr. Powell's report does not explain his methodological framework or even identify specific guidance documents that informed his approach. His "systematic review" lacks the transparency and objectivity that are essential to a scientifically sound review and, as discussed at length in my report, are the heart of my methodology (Rule 26 Expert Report of Brandon Pearson, MS, PhD at 4-7, 66-82); (OECD 2019) ("the important goal of any WoE evaluation is to provide a clear and transparent process that can be easily followed, reproduced, and rationally discussed by all stakeholders").

The scoring rubric I developed for evaluating studies, which was included for purposes of transparently and efficiently conveying my analysis, is criticized in Dr. Powell's report. He suggests that I should have used one of two published scoring systems he cites instead. The first system he references, ToxRTool, was designed specifically for a regulatory context (e.g., Schnieder et al., 2009), which specifically lays out ToxRTool as a system designed to assist regulators in interpreting toxicological data. The current case involves a causality inquiry in the context of product liability litigation, not evaluating data within a regulatory framework. This is the central reason why I developed a methodology based on broad guidance for WoE in the toxicological context and tailored it to the specific developmental neurotoxicity (DNT) parameters relevant to this case. Furthermore, unlike the peer-reviewed guidance documents incorporated into my methodology, the ToxRTool kit is not specifically designed for interpreting DNT data, which features its own unique complexities due to the inherent complexity of neurodevelopment. Similarly, the SciRap tool cited by Dr. Powell "was developed for use *in the regulatory health risk* assessment of chemicals and to promote structured and transparent evaluation of study reliability within European regulatory frameworks" (Waspe et al., 2021) (emphasis added). Regardless, nowhere in his report does Dr. Powell claim that use of either of these other tools would have resulted in a different conclusion. And Dr. Powell himself declined to employ either of these tools or apply any transparent scoring system to his own "systematic review" of the robust literature on the neurodevelopmental impact of in utero APAP exposure.

Furthermore, Dr. Powell's decision to conduct a systematic review and thereby "narrow the evidence" (Powell Report at paragraph 58) marks a clear departure from the WoE approach utilized and, on several occasions, advocated for by the APAP manufacturers and their employees and contractors (APAP-JJCI-0000065365) [redacted] (emphasis added); (APAP-JJCI-0000518712 (Cardno ChemRisk Draft Overview of Toxicological Data and Assessment of Paracetamol (Acetaminophen)) [redacted] (emphasis added); (Dep. [redacted] (May 24, 2023) at 184) ("I believe we always need to look at the totality of the data. And there may be new data that comes out preclinically. There may be new data that comes out epidemiology-wise. There's going to be strengths and weaknesses of all that data that comes out. You need to do a *weight of evidence* assessment of the epidemiology data, the preclinical data, whatever other – all the data that's available, and then make a decision based upon the weight of the evidence") (emphasis added);

(APAP-JJCI-0000007527; APAP-JJCI-0000008526; Dep. [redacted] (June 1, 2023) at 141-143; Dep. [redacted] (May 26, 2023) at 465-466; Dep. [redacted] (May 19, 2023) at 324). Weight of Evidence is the appropriate approach for evaluating a large and diverse DNT dataset (OECD 2007) ("A developmental neurotoxicity study will provide information on the effects of repeated exposure to a substance during in utero and early postnatal development... The interpretation of test results should use a weight of the evidence approach"); (Tyl et al., 2008) ("there are a number of papers that specifically discussed interpretation of developmental neurotoxicity data. All of these previous papers and this paper have used a weight-of-evidence approach." "It should be noted that there are uncertainties, confounders, variabilities, etc., attached to each endpoint evaluated which causes uncertainty around the presence or absence of concordance. Again, a weight-of-evidence approach is strongly recommended to assess the importance to attach to the concordance, or lack thereof, of the effects on other indices within effects").

As I detailed at length in my report, scientific integrity demands that evidence of APAP's neurotoxic effects be considered in context, "as part of a large and well-developed body of scientific work, both epidemiological and experimental" (Pearson Report at 4); (National Research Council (NRC) 2011) ("Clearly, both epidemiology and toxicology have much to offer in elucidating the causal relationship between chemical exposure and disease. These sciences often go hand in hand with assessments of the risks of chemical exposure, without artificial distinctions being drawn between them"); (Tyl et al., 2008) ("The data from these examples are evaluated separately and in isolation for each parameter. In actual practice, however, an evaluation to determine whether there is an indication of a compound-related effect should rely on an integrative evaluation of all the available data in the study (e.g., clinical signs in the offspring and evidence of maternal toxicity), as well as data from other studies (e.g., adult neurotoxicity, reproductive toxicity studies, and other general toxicity studies)"). The NRC's Reference Manual on Scientific Evidence (3d ed.) includes a straightforward explanation of the inextricable link between these two disciplines: "In essence, epidemiological findings of an adverse effect in humans represent a failure of toxicology as a preventive science or of regulatory authorities or other responsible parties in controlling exposure to a hazardous chemical or physical agent. A corollary of the tenet that, depending upon dose, all chemical and physical agents are harmful, is that society depends upon toxicological science to discover these harmful effects and on regulators and responsible parties to prevent human exposure to a harmful level or to ensure that the agent is not produced. Epidemiology is a valuable backup approach that functions to detect failures of primary prevention. The two disciplines complement each other, particularly when the approaches are iterative" (NRC 2011).

However, Dr. Powell's report approaches the question of APAP's developmental neurotoxicity as though the in vivo DNT studies exist in an informational vacuum – devoid of the numerous relevant and well-designed epidemiological studies that consistently demonstrate a causal relationship between in utero APAP exposure and altered neurodevelopment. Further, Dr. Powell completely disregards two more lines of evidence—i.e., in vitro/ex vivo and in silico data—which are undeniably relevant to his inquiry and generally contrary to his ultimate conclusions. Disregarding these lines of evidence may be acceptable in a setting where a drug's safety in

neurodevelopment has already been established, but it is inappropriate where, as here, numerous observational studies indicate that in utero exposure impacts neurodevelopment.

The following sections of this rebuttal report identify and respond to several specific assertions in Dr. Powell's report, with individual paragraphs within his report which I am responding to identified.

Paragraphs 5 and 10. Dr. Powell indicates that studies included in my Weight of Evidence analysis use non-relevant doses of APAP. Contrary to his assertions, my report addresses the clinical relevance of doses within these studies formally throughout. In forming my opinion, I have relied on my expertise as a neurotoxicologist to this end and have fully rationalized the allometric scaling thresholds.

Dr. Powell also argues that the results of the studies within my Weight of Evidence analysis are inconsistent. This criticism is based on unattainable definitions of consistency. This will be addressed and rebutted further later in this report. Dr. Powell also excludes many studies for not correcting for multiple comparisons. This type of correction is not necessary in many or most relatively simple experimental designs. While Dr. Powell states that my report has "lowered the bar" (Powell Report at paragraph 10), this is one of many instances in which he strategically set the bar too high in search of a particular outcome. In stark contrast to the opaque approach used by Dr. Powell, my report includes a thorough explanation of the bases and criteria for inclusion of studies in my Weight of Evidence analysis, and I have further elucidated these bases herein.

Paragraph 9. Dr. Powell incorrectly asserts that a singular directionality of an effect in a study is required in order for that study to be relevant to the overall question of whether it shows perturbed neurodevelopment (Tyl et al., 2008) ("Since different functional domains are evaluated by different tests in the study, a complete concordance of effects is not expected and/or necessary to establish the relevance and/or validity of a finding"). This is another example of setting unrealistic and exclusionary standards for relevant research findings. For instance, Dr. Powell says that some of the cited rodent findings (including work from my lab in Baker et al., 2023) show increased social behavior and that this supports that APAP does not show enhanced risk for ASD. This argument is inaccurate for the following reasons: DSM criteria for ASD indicate that social interactions, including communication, are abnormal or impaired. Consistent with human clinical manifestations of ASD, the DSM does not state that social interactions in people with ASD are uniformly or quantitatively reduced. Likewise, animal models that evaluated ASD-like outcomes must not focus only on reduced social interactions; inappropriate social engagement, even if it is increased, is still relevant. Additionally, findings that are in the opposite direction of the prediction nevertheless demonstrate that the sensitive neurobehavioral system is perturbed by the developmental exposure to the medication. So, while the outcome does not show what Dr. Powell might interpret as directional "face validity" for the ADHD-like or ASD-like outcome, the neurobiological system regulating social behavior is nevertheless reliably affected by the developmental exposure to the medication. A directional concordance is not required. In fact, the article cited by Dr. Powell to support this criticism of my methods, Silverman et al., 2022, indicates that such face validity is not generally the main target or criterion in animal models of ASD or

ASD-like outcomes. In fact, "Statement #1" of their article is entitled "Complete face validity should not be expected to be fully apparent."

Paragraphs 10-11. As noted above, paragraphs 10 and 11 argue that I am "lowering the bar" by including any studies in my WoE analysis that measure any change in the brain or neurodevelopment. This is not the case. I did not include published studies on developmental APAP in rodents that measure pain or analgesia, nor mood, anxiety, aggression, sexual behavior, nor that evaluate stereological differences in sex-specific cellular composition in the brain. Further, as laid out carefully in my report, ASD and ADHD are closely related neurodevelopmental disorders which are highly transdiagnostic involving shared and distinct transcriptional, synaptic, cellular, and neuroanatomical systems. They also have shared and distinct risk factors. In accordance with this complexity, there would not be any one outcome or battery of tests that would solely or uniquely answer the question of whether APAP can cause neurodevelopmental outcomes. Relatedly, even in humans diagnosed with ASD and/or ADHD, each and every type of behavior associated with ASD and/or ADHD is not necessarily present because they are heterogeneous disorders. Similarly in animals, animals may only exhibit one or some of the potential behaviors associated with the ASD/ADHD symptoms modelled in studies. This is the exact reason why evaluating a range of relevant studies in a weight of evidence is appropriate. The studies I included in my analysis were carefully selected because they involve neurochemical, molecular, or behavioral systems that reflect brain systems that have relevance to neurodevelopmental disorders, particularly ASD and ADHD.

Paragraph 35, 77-83. Dr. Powell's report claims that "[f]or scientific findings to be plausible, they must be robust, rigorous, and reproducible." In paragraphs 35 and 77 through 83, he purports to show that studies that evaluated the potential ASD and ADHD effect of prenatal APAP use on offspring evaluated different outcomes leading to multiple discrete findings or evaluated the same or similar outcomes but did not replicate findings. However, he includes studies in his analysis where APAP was administered to adult rodents, while elsewhere claiming such studies have no relevance to whether prenatal use of APAP affects offspring neurodevelopment. Ultimately, while studies in adult rodents may provide high-level insight into APAP's mechanism of action on the body generally, they should not be used to argue that APAP has no effect on neurodevelopment. By their very nature, these studies take place outside the context of neurodevelopmental processes. Using them here to argue results have not been replicated or that studies show different effects is misleading.

Further, alterations of behavioral and non-behavioral outcomes generally have been replicated. Dr. Powell relies on an inappropriately rigid view of replication and concordance of findings in the preclinical APAP literature. It is not to be expected that every study author designs their study in exactly the same way and finds exactly the same thing (Tyl et al., 2008) ("It should be noted that there are uncertainties, confounders, variabilities, etc., attached to each endpoint evaluated which causes uncertainty around the presence or absence of concordance. Again, a weight-of-evidence approach is strongly recommended to assess the importance to attach to the concordance, or lack thereof, of the effects on other indices within effects"). Instead, many studies have their own unique designs, and this diversity adds strength to overall weight of evidence -

which shows that most studies find alterations to locomotion/activity, repetitive behavior, social behavior/communication, cognition, and neurochemistry in response to developmental APAP exposure.

Paragraphs 54-55. Dr. Powell argues in paragraphs 54 and 55 that allometric scaling for preclinical studies of APAP is not appropriate because APAP is not a new drug, and we have decades of data on liver toxicity and analgesia. First, this is a false equivalence. This argument cannot be made for studies of the brain as a target because APAP has been "grandfathered in," as my report indicated, and was never systematically tested for neurodevelopmental safety. This case is not centered on whether APAP causes hepatotoxicity, or other peripheral APAP toxicities, which are well established. Even if those inquiries were central to the question of general causation in this case, it would be important to note that APAP's toxicity continues to be explored as labeling changes to APAP in prescription preparations and other concerns over skin and other adverse outcomes have occurred in the last 10-15 years. Thus, for the brain, allometric scaling is highly appropriate. Dr. Powell also disregards other data points (e.g., CSF APAP levels) when indicating that my comparator levels are super-physiological when, in fact, my report carefully considers concentrations and doses that are relevant and important to considering the neurodevelopmental toxicity of APAP such as toxicokinetic data from Saad et al., 2016, as well as other data points (e.g., additional rat studies) that exceed the in vitro concentrations.

Dr. Powell applies such unduly restrictive criteria that, "[o]f the 99 peer-reviewed, original research publications evaluated in detail, only two publications" met them (Powell report at paragraph 66). Furthermore, both of those studies—i.e., Saad et al., 2016 and Baker et al., 2023—are misinterpreted in Dr. Powell's report, and in the case of Baker et al,. abjectly so, as further detailed below.

Paragraph 68. Dr. Powell provides the following interpretation of Baker et al., 2023 in paragraph 68: "Male pups actually demonstrated increased USVs at one point compared to controls, another finding that is opposite of what would be hypothesized in an animal model for ASD. Thus, the findings of this paper—co-authored by Plaintiffs' expert Dr. Pearson—at best would refute rather than support the hypothesis that acetaminophen leads to behavioral alterations consistent with ADHD or ASD; the study further does not provide a scientifically valid mechanism whereby such a hypothesized effect might occur."

This is an erroneous understanding of what is being measured in ultrasonic vocalizations in rodent pups separated from their mothers. This is not a measure of mouse pups engaging in reciprocal social communication with their mothers or a social partner. In this context, USVs, at a macro level, measure distress associated with separation from their mothers, and at a micro level, dynamics of the development of neural circuitry associated with discomfort, thermal stress, sensation/perception, negative emotion/arousal states associated with the separation/housing/novelty and so forth. As some of the classic literature in this field states: "Reduced *or unusual* USVs in mice may offer a useful assay with reasonable face validity to the second diagnostic symptom of autism, impaired communication." (Scattoni et al., 2008) (emphasis added). Numerous studies of mouse models of ASD have shown increased and altered patterns of developmental dynamics of USVs (e.g., in Shank2 mutant mice) (Ferhat et al., 2016). Dr. Powell's

bias is evident because aside from his misinterpretation of the USV data, he ignores the remainder of the study which includes findings of sex-specific alterations in activity and rearing behavior in the open field test.

Finally, in reference to the Baker et al., study, Dr. Powell indicated that my research group did not provide a scientifically valid mechanism whereby such a hypothesized effect might occur. Meanwhile, at no point in his entire expert report drafted does Dr. Powell acknowledge or engage with the rich RNA-sequencing data that shows sex-shared and sex-specific oxidative stress, hormonal, and metabolic changes in brain tissue of APAP exposed mice relative to controls shown in our study.

Dr. Powell and other defense experts repeatedly state that mechanisms and outcomes my report indicated are not "scientifically valid" whereas my expertise, in fact, sits squarely at the interfaces of toxicity, neuroscience, and behavior, particularly as it relates to etiologies of neurodevelopmental disorders. Merely stating that a result is invalid without appropriate justification does not make it so.

Paragraph 76. Low sample size should not be a criterion to automatically exclude a study from consideration. Studies are capable of revealing meaningful results with or without statistical significance, and statistical significance can be achieved without large sample sizes. Additionally, while behavioral outcomes tend to require large samples sizes, histological and molecular variables may not require as many replicate samples. Accordingly, blanket requirements for sample size cutoffs are inappropriate. While it is critical to consider Type I and Type II error in interpreting data, the blanket exclusion of studies with low sample sizes is another example where the strategic use of exceptionally restrictive criteria serves Dr. Powell's argument by excluding studies that show an effect between APAP and neurodevelopment. The studies Dr. Powell dismisses as irrelevant on this basis are peer-reviewed.

As Dr. Powell is certainly aware, agencies such as the NIH and IACUC (animal equivalent to IRB) require justification of sample size via power calculations. It is routine for peer reviewers to specifically question the sample size of individual studies before a journal accepts them for publication. While it is possible that low sample sizes can rarely, by chance, suggest spurious effects, statistical tests can account for this as p-values are subject to power. Effects seen at lower replicate numbers can reflect robust phenomena. Dr. Powell, in fact, insists that the roughly 13 studies he lists "employ a very low number of animals (range: n = 3-9)" have critical flaws and "do not allow for trustworthy scientific conclusions" because a power calculation must be done before commencing the study. This statement is pure speculation. There is no way that Dr. Powell would know whether those research teams internally performed a sample size calculation or power calculation ahead of publishing their study unless he contacted each and every one of those teams, which he does not indicate having done. Additionally, as mentioned above, histological and biochemical assays tend not to require as many replicates as behavioral studies, and when behavioral effects are robust, smaller sample sizes can yield significant results. Insistence on more replicates than necessary is just as unethical as studies that are performed when underpowered. In summary, Dr. Powell has not provided sufficient justification to exclude these studies.

Paragraph 101. Dr. Powell states: "Because there is no existing biologically plausible mechanism for how acetaminophen exposure—or exposure to any other chemical substance—in utero can cause ASD and ADHD, Plaintiffs' experts offer numerous hypotheses of potential mechanisms. None of these hypothesized mechanisms is supported by actual science." This is an untrue and inflammatory statement. Multiple studies have supported that ASD brain tissue shows higher levels of oxidative stress (Sajdel-Sulkowska et al., 2010; Rossignol & Frye, 2014). Gene transcriptional and epigenetic signatures in ASD biospecimens likewise support involvement of oxidative stress in ASD (Melnyk et al., 2012). These are just a few scientifically sound examples that the preclinical studies confirm.

Paragraph 116. In claiming that my report included publications using a higher concentration of acetaminophen than that considered non-toxic, Dr. Powell misleadingly reports only the human plasma level data I cited. However, my report also provides the rodent plasma and CSF levels resulting from allometrically scaled, non-toxic doses, and the studies I discussed were well within that range. Dr. Powell's mischaracterization of my report here is ironic given that he repeatedly accuses my report of "cherry picking" data.

Paragraph 121. Dr. Powell criticizes my report for giving "little to no consideration to adversity/adaptation, transience/permanence, or primary/secondary toxicities." Dr. Powell has not made a coherent argument here. Dr. Powell has missed multiple places where I discuss direct and indirect toxicity (e.g., Rule 26 Expert Report of Brandon Pearson, MS, PhD at Figure 31). With respect to homeostasis, compensation, and the like, if Dr. Powell would have engaged with the RNA-sequencing data and discussion from the Baker et.al., 2023 paper, it should have been clear to him that detoxification responses versus toxicity responses were considered, and that we also considered transient versus persistent effects.

Paragraph 141. Dr. Powell calls into question the results of Blecharz-Klin et al., 2018 because the 5 mg/kg group showed an effect, but the 15 mg/kg group did not. He uses this to challenge the consistency of social behavior perturbations of APAP. There are various reasons why this could occur. In toxicology and pharmacology, non-monotonic dose responses are not uncommon. Similarly, for this study, higher dose effects could be masked by engagement of NRF2 dependent antioxidant systems that were not engaged at the lower responses, among many other potential explanations. Dose response data can be valuable, but lack of effects at biological gradients or across a dose response is not a parameter that is required for inclusion in a weight of evidence methodology. Insistence on linear dose response effects as evidence of data quality is yet another example of excessive screening that serves to limit research that is damaging to the defense.

Paragraph 142. With respect to Harshaw & Warner, 2022, Dr. Powell states: "using the 3-chamber test of sociability, a widely used measure of preference for a social target versus an inanimate target, acetaminophen alone did not lead to alterations in preference for social target interaction in the acetaminophen-alone group in either sex." Dr. Powell goes on to say "Thus, this publication does not support an effect of acetaminophen administration on repetitive or social behaviors." Dr. Powell selectively addressed a single test and blatantly ignored the significant sex-dependent, and sex independent effects of APAP on anxiety-like behavior (Harshaw & Warner,

2022 at Figure 4A) and social avoidance behavior in the social interaction test (SIT) (Harshaw & Warner, 2022 at Figures 7; 8 A, C, D, F) as well as its significant influence on increased repetitive behavior (Harshaw & Warner, 2022 at Figures 2A, D).

Paragraph 145. Dr. Powell's criticism of my review of the Suda et al., 2021, study ignores that I was fairly critical of this study and the score I assigned it reflected those criticisms. The findings of the study are still relevant to the inquiry at hand. Suda et al., demonstrated that APAP-exposed rats did behave reliably differently in a social arena. Dr. Powell attempts to downplay this with a quick calculation of his own to show that there was an approximately 6% increase in the amount of time rearing associated with the APAP exposure. Even assuming that calculation is correct, it means for every one hour, the rats that were exposed developmentally to APAP spent 4-5 minutes more in an upright behavioral posture, doing things that untreated control rats were not doing. This behavior can be functionally interpreted as scanning for predators, looking for a way to escape (assessing risk), or adopting a defensive upright posture. To put this another way, the form and quality of the dyadic social interaction is disrupted for a significant proportion of the time in these APAP-treated animals. This is a relevant outcome for ASD and ADHD phenotypes – likely both as the quality of the social interaction is quantitatively changed and the exposed animal has their attention elsewhere.

Paragraph 103. Dr. Powell argues that the endocannabinoid system dysfunction as a mechanism linking developmental APAP exposure to neurodevelopmental disorders such as ASD and ADHD are inconsistent with the literature by citing a study where cannabinoids are being tested as therapeutics for ASD. This is a poorly constructed argument. Even if cannabinoids could potentially act as a therapeutic for ASD, their therapeutic role in ADHD is notably not mentioned. This matters not, in the end, because the argument is not cogent. Even if an individual cannabinoid compound were to be effective as a treatment for a neurodevelopmental disorder, it does not follow that that another compound acting on the endocannabinoid system is safe to administer to a developing fetus, nor does it mean that compound does not cause damage. In fact, for the reasons elucidated in my report, disruption caused by chemicals acting on the endocannabinoid system can underly a perturbed developmental cascade leading to a behavioral sequela diagnosable as ASD or ADHD.

All opinions offered in this rebuttal report are held to a reasonable degree of scientific certainty.

Dated: July 28, 2023

Respectfully submitted,

Brandon L. Pearson, Ph.D.